LIGATION OF HEPATIC ARTERIES AND COLLATERAL ARTERIAL CIRCULATION IN DOGS

AUTHOR(S):

ISHIGURO, MINORU

CITATION:

ISHIGURO, MINORU. LIGATION OF HEPATIC ARTERIES AND COLLATERAL ARTERIAL CIRCULATION IN DOGS. 日本外科宝函 1959, 28(8): 2964-2991

ISSUE DATE:

1959-09-01

URL:

http://hdl.handle.net/2433/207007

LIGATION OF HEPATIC ARTERIES AND COLLATERAL ARTERIAL CIRCULATION IN DOGS

by

MINORU ISHIGURO

The 1st Surgical Division, Kyoto University Medical School
(Director : Prof. Dr. CHISATO ARAKI)

〔Received for publication July 29, 1959〕

CONTENTS

I. INTRODUCTION

The greatest of dangers which follow interruption of the hepatic arterial supply is liver necrosis. The more complete is the interruption, the higher becomes the motality of animals operated upon. As has often been reported since the time of

HABERER, especially when the gastroduodenal a. and right gastric a., as well as the common hepatic a., have been ligated, almost all animals die of liver necrosis.

On the other hand, MARKOWITZ, REPPAPORT, and SCOTT reported that postoperative administration of penicillin could remarkably lower down the mortality of animals operated upon. This observation was further confirmed by GRANT, FITTS, RAVDIN and others, and it is now well known that other antibiotics are as effective. However, about 30% of dogs operated upon and treated with antibiotics are reported to have died. Furthermore, LAUFMAN et al and FRASER et al stated that the more extensive and complete was ligation of arteries at the hilus of the liver, the higher was the mortality of dogs···reaching over 50% or even 65% despite antibiotic therapy. According to these reports, all experimental dogs that died as a result of the ligation showed a high degree of liver necrosis, while those which survived did not disclose such a high degree of necrosis long after the antibiotic might well have lost its power. Why was it so? The question is not yet answered. Was it because of the anatomical difference among the animals operated upon? Was it because of incomplete ligation due to some mistakes in the operative techniques? Or because some collateral circulation, which had either existed before ligation or come into being after it, had been substituted for ligated arterise?

In an effort to answer these questions, I made the below experiments in order to investigate the relation between the presence of such collateral arterial circulation to the liver and the development of fatal liver necrosis caused by ligation of hepatic arteries. For this purpose patterns of the liver with extra or intrahepatic circulation system were made by injecting coloured plastics.

II. METHODS OF EXPERIMENT

A. Experimental Materials and Operative Procedures

1) Materials

Healthy adults dogs were employed. Some 60 were injected with coloured plastics, while about 90 were used for histological studies. Besides, a dozen or so were used as the control.

2) Operative procedures

A midline incision of the abdomen was employed under intravenous nembutal anaesthesia. In some cases the common hepatic artery (the c. h. a.), the gastroduodenal artery (the g. d. a.) and the right gastric artery (the r. g. a.) were doubly clamped, ligated and severed, while in others the other arterial branches (which will be mentioned later) together with the above-stated 3 arteries were subjected to the same operation either all at the same time or in two different stages. (The abbreviations of each of the 3 arteries are given in the brackets respectively.)

After the operation, 100, 000 or 300,000 units of penicillin were given to the dogs once or three times. Those which survived were subjected to the below experiment of injection of coloured plastics. For the sake of contrast, both those dogs which died of liver necrosis in spite of administration of penicillin, and those with ligation of only some of the above mentioned 3 arteries were also examined.

B. Experimental Method

1) Injection of India ink

Before injecting plastics into the vascular systems, India ink was injected in order to make the observation more exactly.

Dogs which survived the operation were anesthetized with intravenous nembutal and subjected to median laparotomy. A ligature was then placed around the stem of the portal vein (the p. v.) at the hilus of the liver, and another ligature around the inferior vena cava (the i. v. c.) just below the liver. Then left thoracotomy was performed either under positive pressure by means of intratracheal intubation or sometimes under normal pressure. No sooner had drop by drop injection of India ink begun than the stem of the p. v. and the i. v. c., now ready for ligation, were tied up. By this method, it was expected, the India ink would be prevented from flowing into the liver through any other route than the arterial vessels.

The injection was continued until the injected material filled capillary vessels on the walls of abdominal viscera, dyeing it black. Then all length of the digestive tracts from the oesophagus to the rectum and retroperitoneal organs were taken out with the diaphragm and principal vessels. The filling ink was traced in order to observe how extrahepatic arteries coursed into the liver. After obtaining the materials for histological studies, injection of plastics was performed.

2) Injection of plastics

a) Irrigation

In order to inject the plastics easily and exactly, the vessels of the resected organs were irrigated by clear water put in from the cut end of the thoracic aorta under the pressure of about 100 mmHg. The portal circulation system and sometimes hepatic veins were also irrigated with some modifications·· in the former case water was injected from the hilus of the liver under the pressure of about 15 mmHg, and in the latter irrigation was done retrogradely from the v. c. under much the same pressure.

The instrument of Dr. Ikegami at Kyoto Univ., with some inprovements on it, was used for the procedure of irrigation. It was continued till grossly there was no blood seen in the water flowing out of the hepatic vein. Much care was taken to keep the pressure of running water at so physiologic a level as not to wash away any obstructions caused by thrombus or some other factors which had existed when the dog was alive, but as to wash away those obstructions caused by postmortem coagulation and so on.

b) Injection of plastics

The techniques of Dr. Ikegami were chiefly followed in preparing and injecting plastics. First, intermediate methyl methacrylate polymer, especially prepared for the purpose, was diluted with monomeric methyl methacrylate. Next, to this mixture were added dye-stuffs and benzoyl peroxide, a stimulant of plymerization. Then, this fluid, kept in a comparatively low degree of viscosity, was injected successively into the common bile duct, the aorta, the p. v., and the i. v. c. from the same portions as when the irrigation was done. No special instrument but a cryster pipe was

used for the injection. Care was taken not to make injection pressure too high. Just as much of the fluid, and not more than that, was injected as could both fill up capillary vessels running over the wall of the gall bladder, and be barely seen through the liver capsule.

As for the volum capacity of intrahepatic vascular systems, the arterial system is the smallest, then comes the portal one. In order to investigate collateral arterial pathways, however, a much larger amount was injected into the arterial system than its capacity, because there was a possibility of flowing into the other organs.

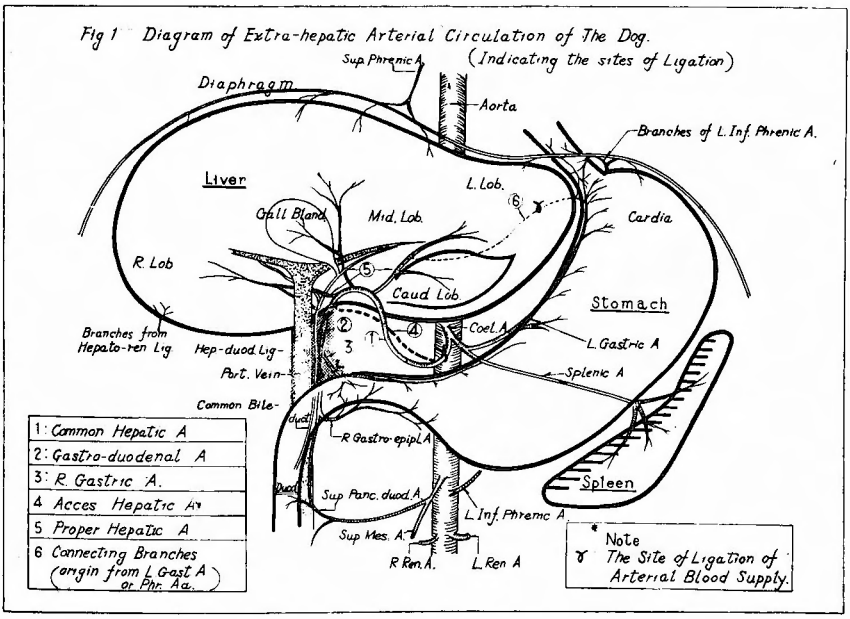
For dyeing matters was used fine powder of pigment, which was mixed with the plastics just before injection, and sometimes oilcolour or thickened lacqure was also used. As the size of particles differs in different dyes, the speed of polymerization varies according to what dye is to be added. Red was chosen for the artery, blue for the p. v., yellow for the vein and white for the bile duct.

c) Corrosion of tissues, washing and observation

After the injection of plastics, the injected liver was immerted in running water for about 24 hours so as to prevent destruction of its vascular systems by the heat of polymerization. After the polymerization had been completed and materials for histological examination had been obtained, the liver was dipped in 30. to 40% caustic soda solution, left for 2 to 3 days in and incubator kept at 37°C in order to corrode unnecessary tissue, which was then washed off by water. In this way, I got the vascular pattern of the liver with that of its adjacent organs.

Observation was carried on these patterns grossly or using the magnifying glass, while the sections of the liver was examined histologically.

III. RESULTS OF EXPERIMENT



A. Findings of Control Group

1) Arterial System of Normal Dogs

The moment India ink was injected into the thoracic aorta of the dogs which had not been operated upon, the liver, as well as the other abdominal viscera, turned black. Microscopic observation revealed that arterial capillaries in the connective tissue and sinusoids were almost pervaded with the injected India ink.

The injection of plastics presented a clear picture of extra and intrahepatic arteries and their mutual anatomical relations.

a) Chief arteries to liver

There are three chief routes to the liver which arterial flow takes; one is from the c. h. a. to the proper hepatic arteries (the p. h. aa.); another is from the superior mesenteric a. to the liver through the sup. pancreaticoduodenal a. and then the g. d. a., if the direction of blood flow is put out of the question; and the third is from the gastric coronary a. to the liver through the r. g. a. (Fig. 1). It is well known, as URABE in our clinic has minutely reported, that when these three principal hepatic arteries (referred to as the hep. 3 aa. in the following pages) have been ligated and severed, most of the arterial blood supply to the liver is cut off, and that nearly all of the dogs which received no antibiotics die of liver necrosis.

b) Intrahepatic vascular systems, and their mutual relations

Only points of importance are given here. In the dogs, the hep. a. starts on its course from the aorta, and ascends toward the hilus of the liver, passing a little left and inferior to the stem of the p. v.. Thereabouts the artery forms 'an arterial arch' where it changes from the c. h. a.. to the g. d. a. From this 'arterial arch' arise the p. h. aa.---first, the right main branch ramifies, then the left one, from

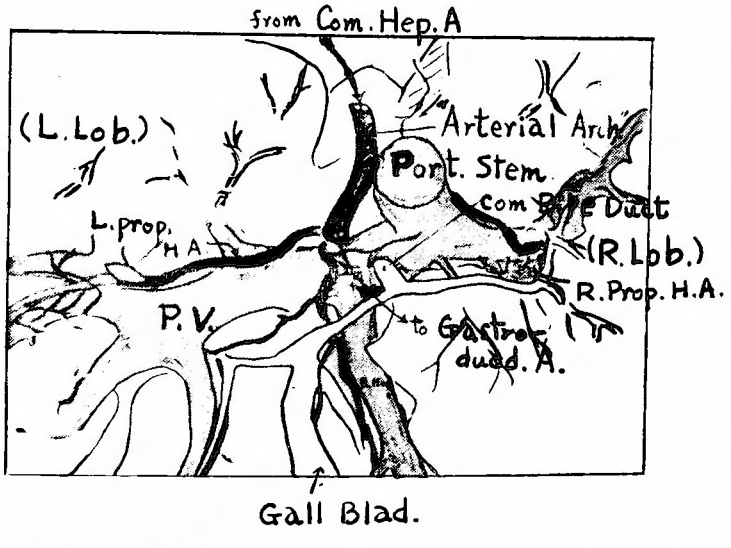


Fig. 2 (cf. page 2981) Branches of proper hepatic a. ramifying at hilus of liver in normal dog, and their relation with portal venous system.

which in most cases the middle one comes out. These enter the liver and supply its various lobes, giving off small branches which course always with the corresponding branches of the p. v., now running along them, now much like a vine on a tree (Fig. 2).

2) Cases in which Common Hepatic Artery Was Solely Ligated

In this case the dogs were able to survive the ligation without antibiotics. The India ink or plastics injected into the aorta was observed to have flown into the liver by way of the g. d. a. just as in normal dogs (Fig. 3). It may safely be assumed that the arterial blood supply to the liver was available by reverse flow through the g. d. a.. By the way, in this case no marked enlargement of the r. g. a. was observed.

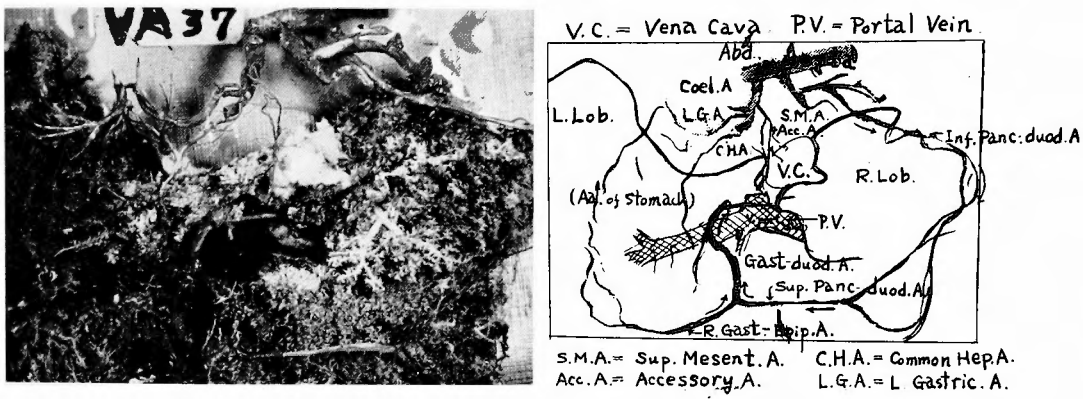


Fig. 3 Extrahepatic arterial circulation after ligation of only common hepatic a. (Arterial blood entering liver chiefly by way of gastroduodenal a.)

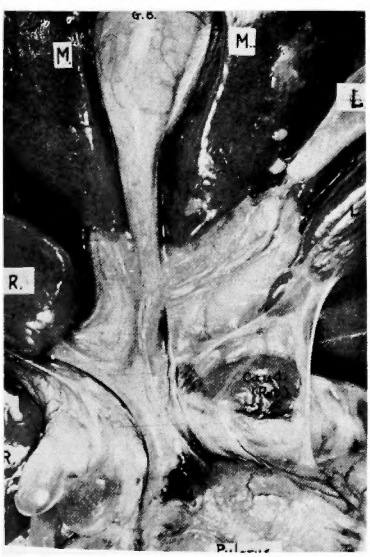


Fig. 4 Capillary arterioles in hepatoduodenal ligament of normal dog.

3) Collateral arterial circulation

What collateral circulation to the liver exists, except the hep. 3 aa.? To what degree could it work as a substitute for them? To answer these questions, the hep. 3 aa. ligated, and then India ink or plastics were injected by way of the aorta. The findings obtained are as follows:

a) Collaterals inferior to liver;

i) The 'accessory artery' which arises from the c. h. a. close to the coeliac axis, passing through lymph glands that swarm around the c. h. a. or the p. v., and enters the liver···especially its right lobe.

ii) Those small arterial twigs which branch off chiefly from gastric or pancreatic vessels and course upwards in the hepatoduodenal ligament to the liver. (Fig. 4)

iii) A network of capillary vessels which

spread along the wall of the common bile duct or that of the p. v. to liver, and which are thought to be nutrient vessels to these walls.

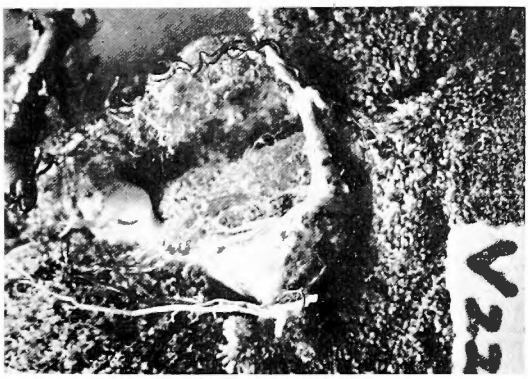


Fig. 5 Collateral arterioles ramifying over diaphragmatic liver surface only, chiefly originated from phrenic aa. to spread only very superficially, so that none are seen to enter liver deeply.

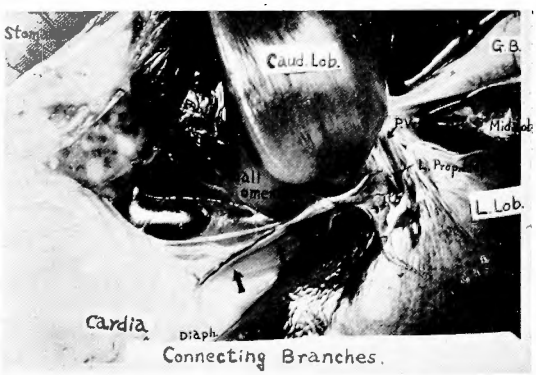


Fig. 6 Connecting arterial branches arising from left gastric a. to course through cardiac part of the stomach of normal dog. Injected with India ink.

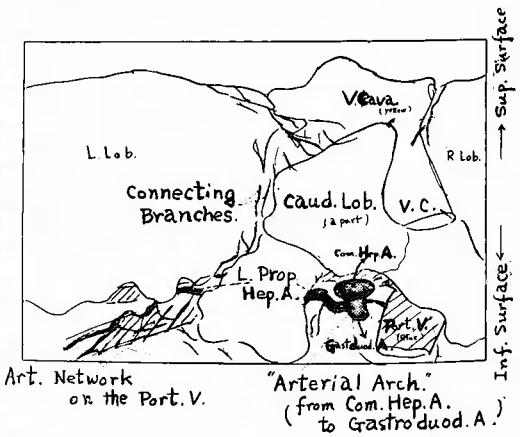


Fig. 6' Same as the case of Fig. 6, but injected with plastics.

iv) Vessels which seen to nourish the wall of the i. v. c., and very few similar filamentous vessels coursing through the thin film between the liver and the right kidney (the hepatorenal ligament), both reaching and spreading over the capsule of the right inferior liver lobe.

b) Collaterals on the diaphragmatic surface of liver;

i) Most of those, who attribute survival of dogs with ligation of the hep. 3 aa. to a postoperative collateral arterial supply to the liver, point out that those passages to the liver come from the phrenic aa. (the phr. aa.). To investigate this point, therefore, especially strict observation was performed. No twigs, however, were found arising from the phr. aa. directly to the liver, except those few very thin vessels which spread over only localized part of the liver surface to which the falciform ligament attached. (Fig. 5)

ii) However, the following collateral arterial circulation, more effective than the above, was observed in all dogs: at the cardiac part of the stomach and the oesophageal foramen of the diaphragm, branches of the left gastric a. anastomose with those of the phr. aa.. A couple of these branches connect with the left main branch of the p. h. aa., after coursing in the small omentum, alongside the liver and between the caudate (reticule) and the left inf. lobes (Figs. 6 & 6'). These collaterals, however, are so thin that they cannot possibly be considered enough to feed the whole liver.

Table. 1 Summary of Cases, Injected with Plastics.

Ligation methods / Survival or Death / Time after ligation	Penicillin treated: Ligation of Hepatic 3 Aa. Died	Penicillin treated: Ligation of Hepatic 3 Aa. Killed	Penicillin treated: Ligation of Hepatic 4 Aa. Died	Penicillin treated: Ligation of Hepatic 4 Aa. Killed	Penicillin treated: 2 Stage Ligation of Hep. 4 Aa. Killed	Penicillin treated: Ligat. of Hep. 2 Aa. Died	Penicillin untreated: Ligat. of only Com. Hep. A. Killed	Penicillin untreated: Ligat. of Hep. 3 Aa. Died	Penicillin untreated: Ligation with other Methods Killed	Penicillin untreated: Ligation with other Methods Dies	Contrast: 1) Normal Dogs (without any Operat.)
within 3hs								V_{101} V_{102}			V_{17}
3~15hs											V_{26}
15~24hs	V_{16}										V_{80}
24~48hs						V_{69} V_{70}		V_{39}		V_{64}	V_{81}
48~72hs	V_{13} V_{14}					V_{35}		V_{9}			V_{82} and 8 Cases
3~ 4ds	V_{30}		V_{71}								
4~ 7ds	V_{11}									V_{38}	2) Nomal Dogs (immediately after ligation of Hep. 3 Aa.
7~10ds		V_{22}		V_{74}							
10~14ds	V_{02}	V_5 V_{19}	–		V_{114}				V_{37}		V_{28}
14~17ds		V_{10} V_{20}		V_{60} V_{120}	V_{40}						V_{29}
17~21ds		V_{21} V_{36}			V_{75} V_{76}						and 4 Cases
21~30ds		V_{18} V_{31} V_{46}									3) others
1~ 3ms		V_6									
3~ 6ms							V_{13}				V_{43} V_{92}
over 6ms		V_{01}					V_{15}				and 4 Cases
Number of Cases	6	12	1	3					1	2	Total
	18		4		4	3	2	4	3		63

4) Anatomical variations in the principal aa

Such variations are seldom found, or if any, they could be easily noticed at the time of operation.

B. Cases which Survived Ligation of Hepatic Arteries due to Administration of Antibiotics

1) Ligation of Hepatic. 3 Arteries

First, a brief comment is given below on each case subjected to this method of ligation; (Table 1)

Case 1 (V_{19}): After the ligation, this case appeared obviously likely to live long, so it was sacrificed on the 11th day. Grossly, the liver was nearly normal. The tip of the middle lobe and its adjacent portions were observed to be still congested; both the sup. and inf. surfaces of the liver were adherent to their respective neighbouring viscera. The gall bladder was found to be perforated.

In the plastic pattern of this case, several comparatively thin arterial branches pass through hep. lymph glands to enter chiefly the left sup. and inf. and the right liver lobes. These branches, however, scarecely found their way into a part of the middle lobe and the quadrate lobe, where most of the branches of the portal vein were not accompanied by arterial ones. In spite of the absence of arterial branches, these lobes showed no more marked histological changes than the other ones.

Case 2 (V_{22}), Case 3 (V_{21}); These cases were killed on the 10th and the 17th post-operative day, respectively. In both, a little cicatricial changes were found in the edges of the left inf., middle, and quadrate lobes, with perforation in the gall bladder of the latter case. Their plastic patterns revealed arteries, only thinner than normal ones, at the hilus of the liver. These arteries were seen to be connected with the coeliac axis by means of a single thin arteral route perhaps the accessory a.. It may be assumed that arterial blood supply to the liver was being made through this accessory a., but with perforation found in the gall bladder. It is doubtful whether this single collateral could supply blood enough for survival of the dogs immediately after the ligation.

It may be added that in none of the 3 cases any arterioles were found arising from the phr. aa. to ramify into the liver and that histological examination revealed a medium degree of local lesions due to the ligation of the hep. 3 aa.…central atrophy of hep. cell cords, entailing enlargement of perisinusoidal spaces and that of cystic capillary vessels in the connective tissue.

Case 4 (V_{36}); This cases was sacrificed 19 days after the ligation. The gall bladder had already been perforated. The ligation of the hep. 3 aa. was found to have been perfectly performed, but a couple of arterioles in the hepatoduodenal ligament were observed to enter the liver. In addition, between arteriolar network at the cardiac part of the stomach and the p. h. aa., there was found a connection, through which an arterial supply, though less than normal, had been distributed as far as the liver edges (Fig. 7).

Histologically, the greater part of the liver was normal, although in some portions liver cells had been degenerated to a medium degree, with sinusoids still congested. The extent of degeneration and congestion, however, seemed to be reversible.

Case 5 (V_{20}); This is a case in which ligation remained more effective. When it was killed on the 17th day after the ligation, both the sup. and inf. surfaces of the liver were found comparatively closely adhered to neighbouring viscera with little inflow of the injected India ink.

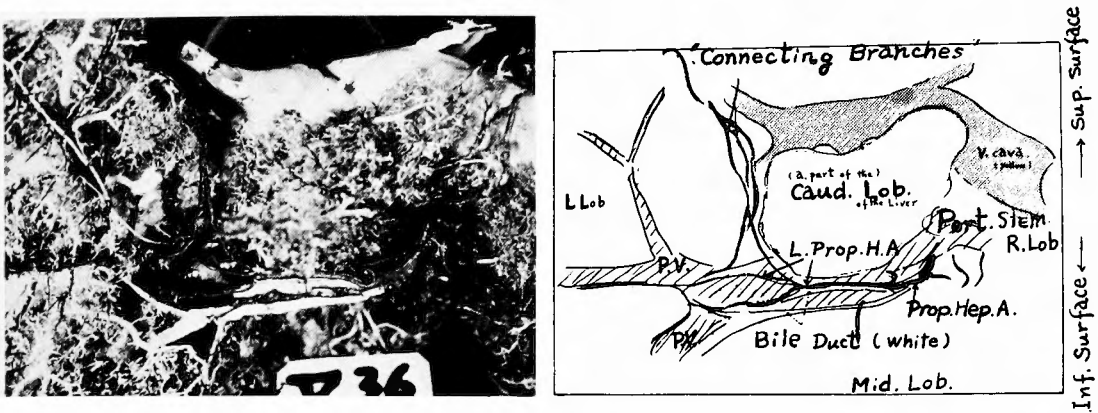


Fig. 7 Intrahepatic ramification of connective branches from left gastric a. (or phrenic aa.) a. seen much thinner than in normal case. (Case 4-V_{36} with ligation of hepatic3 aa.)

The plastic pattern revealed only a few filamentous arterioles, now replaced by the injected red plastic, at the hilus of the liver. These are identified to be small, yet slightly enlarged, arterial branches coursing along the hepatoduodenal ligament. On tracing them, it was found that most of them spread only over the liver capsule or ligament at the hilus of the liver, but one or two of them coursed along the stem of the p. v. and entered some part of the liver parenchym of each lobe together with the main branch of the p. v. (Fig. 8). They were all very thin and went winding.

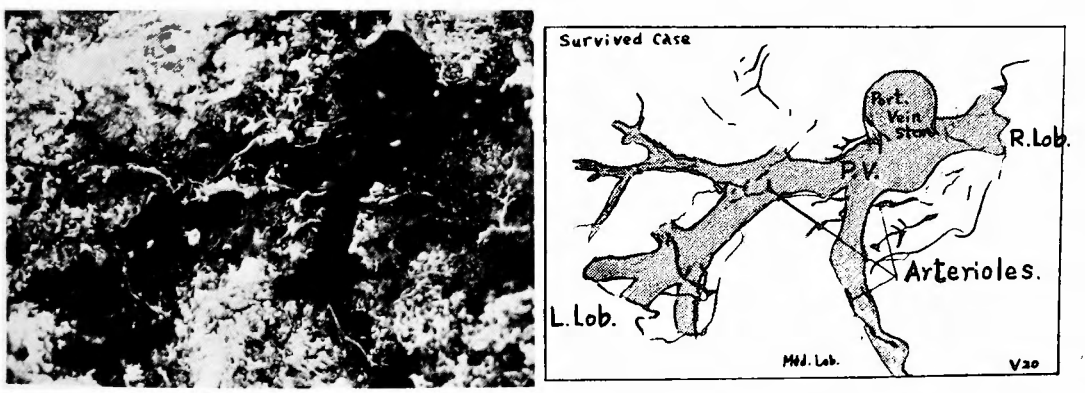


Fig. 8 Arterioles ramifying in hepatoduodenal ligament, much thinner and more irregular than normal. (Case 5-V_{20})

As for the surface of the liver facing the diaphragm, no intrahepatic arterial ramification was observed to originate in the phr. aa., except only a few very thin arteioles seen on that portion of the liver surface close to the i. v. c. and cardiac part of the stomach. When followed up, some of these entered the subcapsular portion of the left liver lobe, where they gave off branches only locally, while others coursed between the left and the caudate lobes to anastomose with the above-mentioned thin arterioles coming from the hilus of the liver.

In addition to the above collaterals, the plastic pattern showed some capillaries on those portions of the liver surface which were adherent to adjacent viscera, but they were very thin and did not enter the liver.

Histologically, the greater part of the liver was normal, with only central atrophy of hepatic cell cords in a part of the liver edges.

Case 6 (V_{18}); In this case, killed on the 24th postoperative day, the branches arising at the cardiac part of the stomach were observed to have become thicker and connected with the existing left main branch of the p. h. aa.. However, these branches were yet powerless, so that they lingered only at the hilus of the liver and the caudate lobe, and did not go so far as the liver edges.

Histologically, the greater part of the liver was normal, except very small area where parenchymal cells were still swollen and edematous, and sinusoids congested, which proves that in these parts oxidation had not yet returned normal.

Case 7 (V_{46}); In the case which was sacrificed on the 28th day after ligation, such connective branches as mentioned above were found thicker than those in Case 6, though weaker than normal, spreading over and reaching the edges of the liver lobes (Fig. 9). The gall bladder was found to be perforated in this case, too.

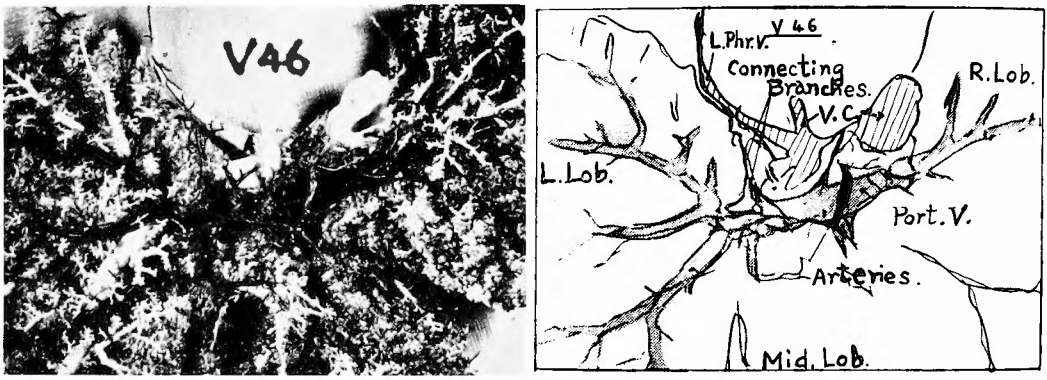


Fig. 9 Intrahepatic ayterial ramification by connective branches from near the cardiac part of stomach, having become stronger than in other cases, but weaker than normal. (Case7-V_{46})

The following cases survived longer than the above:

Case 8 (V_6); This case showed defferent finding from the other ones. When it was sacrificed 42days after the ligation, the liver lobes were found comparatively closely adherent to each other and to the diaphragm. There remained rudimentally what seemed to have

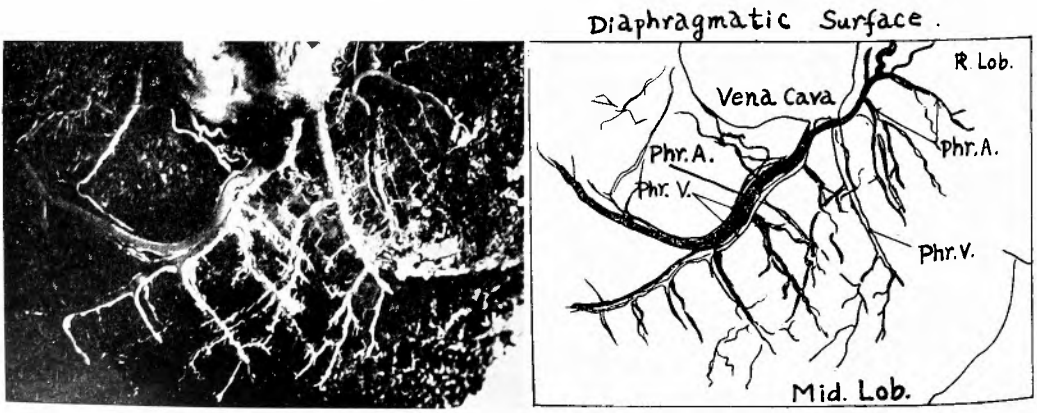


Fig. 10 Enlarged branches of phrenic aa. (Case 8-V_6 with ligation of hep. 3 aa.).

been a gall bladder. The liver parenchyma, however, suffered no abnormality worthy of mention. Histologically, no changes were observed in the liver cells, either.

Now for another purpose, plastics were injected into the liver from the peripheral cut end of the. c. h. a.. As had been expected, the injected material deffused over all liver lobes systematically and went unexpectedly, farther to the diaphragm. Moreover, these branches were found winding and so much enlarged that they were thicker than the phrenic veins (Fig. 10).

In this case, as the liver had not been taken out with the aorta, the plastics injected through the c. h. a. are assumed to have come to the phr. aa., not by way of the aorta, but through some direct connectives made between the branches of the hep. aa. and those of the phr. aa.. When one of these winding branches of the phr. aa. was really traced, it took an unusual route between the left and middle lobes and connecting with the left main branch of the p. h. a.

However, this was no thicker than about 1/25 of the stem of the p. v..

Case 9 (V_{01}); In this case which survived the ligation than a year, any injected material was not found diffusing in the hepatic vascular system by the injection of India ink.

Such were the cases with the other cases.

The above 12 cases survived ligation of the c. h. a., the g. d. a., and the r. g. a. under the administration of antibiotics, and were then sacrificed 10 days to more than a year after the operation. All of them were not completely deprived of arterial blood supply to the liver, though the extent of the deprivation varied from case to case. In some cases arterial blood entered the liver through those small vessels which passed through lymph glands swarming around the c. h. a. or the p. v.; in some, through a little enlarged vessels in the hepatoduodenal ligament; and in more others, through those slightly widened branches passing the cardiac part of the stomach to anstomose with the l. p. h. a.. All these collateral pathways, however, were by far weaker than normal. Except them, only newly vascularized capillary vessels were seen entering the liver through adhesions caused by the operation, only to ramify on some portions of the liver surface. However, they did not course with the branches of the p. v., but spread only superficially over some parts of one lobe of the liver.

In case of a lobular liver like the dog's, in order to supply each and every one of the lobes systematically, it is necessary for arterial blood to enter the liver at its hilus. Therefore, any arterial collaterals entering the liver at any place other than the hilus may supply only a small portion of a single lobe, if they are not connected with any arteries coming in at the liver hilus. On the contrary, if and when they are anastomosed with any arteries entering the liver at its hilus, then they can possibly supply the whole liver. Thus, except the hep. 3 aa., it is the very connective branches coming from the cardiac part of the stomach that count for the most as collateral passages to the liver. However, in Cases 1 to 5, to say nothing of normal ones, such collaterals appeared too thin to carry arterial blood enough to supply the whole liver. In cases which survived longer, it was expected that these collaterals would be so enlarged as to enable an ample arterial blood supply to the liver. The expectation was realized in Cases 6 and 7, where such collateral pathways

had been gradually widened until about a month after ligation they carried a considerably large amount of arterial blood to the liver. It may be interesting to note, however, that when sacrificed these two cases had already had perforations in the gall bladder. The gall bladder is nourished with arterial blood chiefly through the cystic artery ramifying from the middle main branch of the p. h. a.. URABE in our clinic has reported that 44% of those cases which survived ligation of the hep. 3 aa. with the help of antibiotics showed perforations in the gall bladder after the 7th postoperative day. As stated above, the two cases of mine had also perforations in the gall bladder. This may well be because very little arterial blood supply to the gall bladder and accordingly to the whole liver, was available at least during a short period after the ligation. In other words, the connective arterial branches of cases 6 and 7 arising from the cardiac part of the stomach had not been enlarged at the beginning; during the early, short postoperative stage, which is so decisive of the fate of dogs operated on, these collateral arteries are thought to have been ineffective in preventing the liver from developing massive necrosis.

Now a few lines for another collateral circulation. Except in a single case, no branches were seen coming from the phr. aa. directly into the liver. The exception was case 8 which had some branches connecting the enlarged phr. aa. with the p. h. aa.. This case may suggest a probability that as late as one and a half months after ligation, there would be some collaterals, enough to nourish the liver, arising from the phr. aa. to enter the liver by some way or other, and spread over a part or sometimes a greater part of it. However, this does not count for so much in the present study, for the problem we are here especially concerned with is not the circulating state of intra- and extrahepatic arterial system as it stands long after ligation, but the presence or absence of effective arterial collaterals at the early, short and decisive stage of postoperative course···decisive on the occurrence of liver necrosis.

On the other hand, about 54 % of those dogs I operated upon died of liver necrosis. This figure marks the lower rate of survival than those heretofore reported (about 30%). This may be because, compared with the previous experiments made by many predecessors, in my study the hep. aa. were ligated as extensively as possible, with an added resection of neighbouring tissues, so that greater lesions were brought about, resulting in death of more dogs operated on.

2) Ligation of Hepatic 4 Arteries

Seven dogs were operated upon; not only the hep. 3 aa. but also those connecting branches, apparently effective collaterals, which course from the cardiac part of the stomach to the left main branch of the p. h. aa., were ligated. (This procedure of ligation will be referred to as ligation of the hep. 4 aa. in the following pages.) Among others, the c. h. a. was as extensively ligated as possible with its adjacent lymphatic system. The result was: one died of liver necrosis the next day; three died 3 to 4 days after ligation; and only the other three survived. If it is permitted to speak in terms of percentage, with this limited number of experimental dogs, the mortality was 57 %, while in the above cases (with ligation of the

hep. 3 aa., it was 54%.) This means that the addition of these collateral pathways to the hep. 3 aa. to be ligated did not bring about a very marked increase in the mortality.

Survival case 1 (V_{120}); This case was killed on the 16th postoperative day. The greater part of the liver was found normal. Only in those portions which are easy to get necrotic, such as the tips of the middle and quadrate lobes, the peripheral parts of the left lobe and the caudate (reticule) lobe, there was recognized somewhat cicatrized changes suggesting that they had once been necrotic. In the plastic pattern, several small branches arose directly from arteries close to the pyloric part of the stomach and near the region of the pancreas-head to stretch out to the hilus of the liver through the hepatoduodenal ligament, giving off smaller twigs. Some of them went as far as the liver edges, becoming thinner as they went together with branches of the p. v..

Case 2 (V_{60}); killed on the 16th day after ligation. In this case, too, what seemed to be small branches in the hepatoduodenal ligament were found a little thicker than in Case 1, reaching the hilus of the liver. It was impossible, however, to trace them to the liver edges (Fig. 11). This fact means that the dog had survived the ligation of the hep. 4 aa. without any effective intrahepatic arterial branches. What is more, the histological findings were normal in the greater part of the liver, although central atrophy of hepatic cell cords and enlargement of perisinusoidal spaces were observed in some small areas (Fig. 11′). In

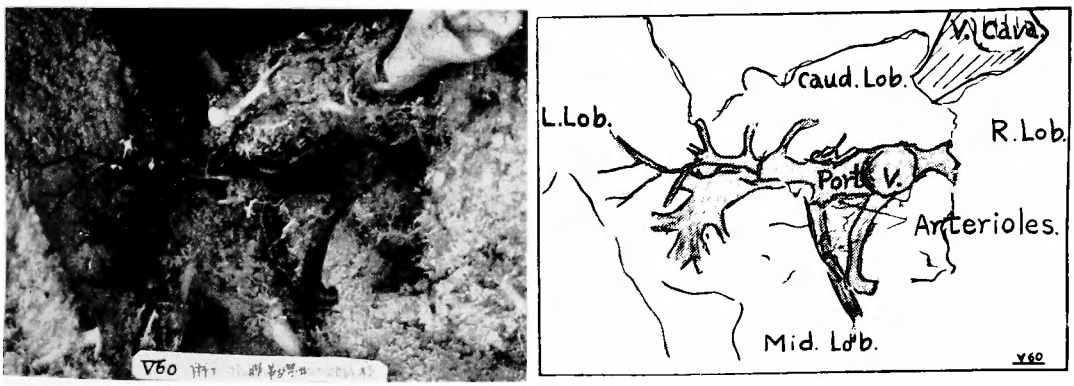


Fig. 11 Arterial branches remaining in hepatoduodenal ligament. (Case 2-V_{60} which survived long after ligation, without intrahepatic ramification effective aa..

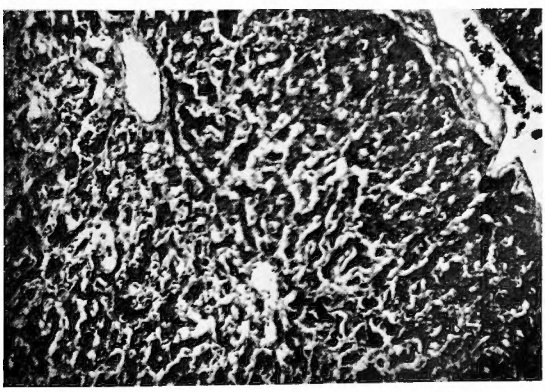


Fig. 11′ Histological picture of the above, stained by H-E (×100). Slight degree of central atrophy of hepatic cell cords and enlargement of perisinusoidal spaces seen only in very small portions of liver.

neither of the two cases, any branches were seen originating directly from the phr. aa..

Case 3 (V_{74}); This case, sacrificed on the 10th postoperative day, revealed much the same histological picture as the above two whereas the plastic pattern showed that at the hilus of the liver there gathered small arterial branches, a couple of which anastomosed with branches of the p. h. aa. and entered the liver. But compared with normal ones, they were filamentous.

As is shown above, even by ligation of the hep. 4 aa. it was very difficult to deprive the liver completely of all its arterial supply, although in one survival case the deprivation was more successful…the liver received less arterial blood. A more extensive ligation would result in nothing but an increase in the mortality because it would cause greater lesions, so the ligation was carried on at two different stages.

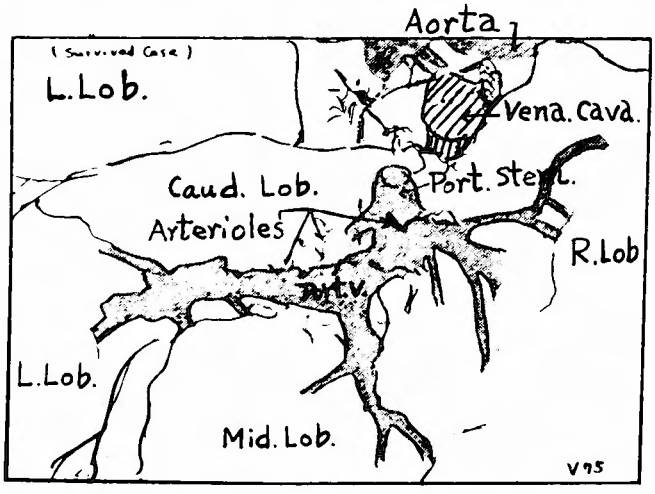


Fig. 12 (cf. page 2981) Case 3-V_{75} with 2-stage ligation. Just as in Fig. 11, this case survived long in spite of the existence or scarcely any intrahepatic ramification of effective aa. Only a couple of thin branches seen running to liver through hepatoduodenal ligament, only to fail to enter liver

Fig. 12' Histological picture of the case-V_{75} (Fig. 12,) stained by H-E (×100). Tendency to regeneration of hepatic cells are seen. All the above cases are either normal ones or ones which survived ligation.

3) 2-Stage (Extensive) Ligation of Hepatic 4 Arteries

The purposes of the experiment will be described later in detail. Those dogs, which survived the lst stage ligation of c. h. a. and the g. d. a., were submitted to the 2nd stage ligation of both the r. g. a. and those connecting branches coming from the cardiac part of the stomach, in addition to cutting all the hepatoduodenal ligament except the p. v. and the common bile duct. Four dogs were operated on and all of them survived due to postoperative administration of penicillin.

Case 1 (V_{114}); This was reoperated upon 9 days after the lst stage ligation. After ll more days it was sacrificed. At the time of reoperation, adhesions had already been so strong that the 2nd stage ligation could not be well performed.

Case 2 (V_{40}); In the light of the failure in the above case, this was reoperated on the 48-th hour after the lst ligation, when a considerably high degree of changes were observed in the liver edges. The dog, however, could well survive the 2nd stage ligation until it was sacrificed 15 days later. Postmortem examination revealed a greater part of the liver to appear quite normal, with perforations in the gall bladder. Nevertheless, the plastic pattern showed no regular intrahepatic ramification of arterial branches.

Histological examination, too, presented a normal picture of nearly all portions of the liver, except in the edges, which had suffered some changes as a result of the lst stage ligation, where there remained a slight degree of changes uncured, although regeneration of parenchymal cells was observed along the border of necrotic area.

Case 3 (V_{75}); This case was subjected to the 2nd stage ligation 4 days after the lst. 17 more days later it was sacrificed. Plastic injection revealed no systematic ramification of intrahepatic arterial branches but a couple of small branches remaining in the hepatoduodenal ligament. (Fig. 12) Histologically, nor marked changes were found, much less degeneration of hepatic cells. (Fig. 12')

Case 4 (V_{76}); This was reoperated on the 6th day after the lst ligation, and 20 days later it was sacrificed. By the plastic injection only a few irregular thin arterial branches were observed close to the hilus of the liver, with no systematic ramification of arterial branches reaching the liver edges. On the other hand, histological study presented a picture of desturbed rows of liver cell cords and a tendency to congestion only at the edges of the liver, where a tendency to regeneration was also observed.

4) Comment.

Of the above 19 cases, on which observation was made, several suffered no serious damage to the liver with a good prospect of long survival, in spite of there being scarcely any intrahepatic arterial branches.

Complete interruption of all arterial blood supply to the liver is very difficult to accomplish, as many predecessors have said. In the present experiment, some thin arterial branches from the hilus of the liver were found in most cases by injection sometimes spreading over each liver lobe to the very edge, though weaker than normal. Such was especially the case with ligation of the hep. 3 aa.. It is highly probable, however, that although immediately after the ligation such arterial branches were either very few or weak, they had increaased or become stronger by the time of injection of plastics. On the other hand, if antibiotics were not given, (nearly) all dogs died even with ligation of the hep. 3 aa. a method of ligation not enough to interrupt the whole arterial blood supply to the liver. From this, too,

it may be concluded that these remaining thin arterial branches are not the only reason for survival of the dogs operated on, and that the liver could survive without effective arteries.

Indeed, these collaterals may have been weak and ineffective at and after ligation of the hep. aa., but were they utterly meaningless? Wasn't it partly because of their presence that the operated dogs could survive? Without these thin collaterals, could even antibiotics have prevented the dogs from dying of massive liver necrosis? To answer these questions, the following experiment was made.

C. Cases which Died of Liver Necrosis in Spite of Antibiotic Therapy

1) After Ligation of Hepatic 3 Arteries

India ink or plastics were injected into several cases from the aorta. As in the above 19 cases which survived, all dogs that died showed a trace of small postoperative arterial blood supply to the liver. To take one remarkable case for example, the dog (V_{30}) which died of liver necrosis 4 days after the ligation of the hep. 3aa. revealed a systematic ramification of intrahepatic arterial branches which carried quite an amount of arterial blood to the liver edges. (Fig. 13) Moreover, histologically a high degree of central necrosis was observed almost everywhere in the liver (Fig. 13′).

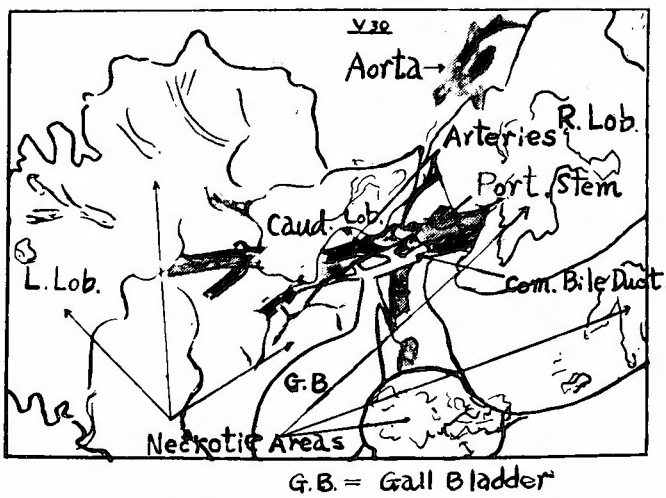


Fig. 13 (cf. page 2981) Case-V_{30} with ligation of hep. 3 aa. This died of massive liver necrosis in spite of postoperative antibiotic treatment and remains of considerably effective intrahepatic arterial branches.

It may not be proper to say that the plastic pattern as it stands shows a real picture of the state of the hepatic circulation but some cases died of liver necroses in spite of the fact that they had much the same anatomy of intrahepatic arterial branches as that of the cases which survived. This may be ample proof that when the hep. 3. aa. have been ligated, a couple of collateral arteries which have been overlooked at the time of opration have nothing to do with prevention of liver necrosis or death of dogs operated on. To investigate this point further the following experiment was made.

2

12

13

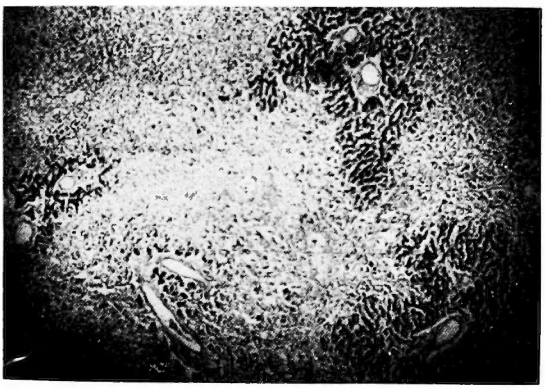


Fig. 13' Histological picture of the above, Case-V_{30} (Fig. 13), showing massive necrosis. stained by H-E (×60).

2) Ligation of Hepatic 2 Arteries

In this experiment, two of the hep. 3 aa. were ligated, while the other intentionally left intact. As was previously mentioned, when the c. h. a. has been ligated, the arterial blood supply to the liver is chiefly done by way of the g. d. a. This fact has often been reported by many experimenters. The addition of the r. g. a., the weakest of the hep. 3 aa., to the above to be ligated would make no difference in the outcome; in fact, those dogs which had been subjected to this methods of ligation survived with the help of antibiotics. So the c. h. a. and the g. d. a. were both ligated, leaving the r. g. a. as it was. (This way of ligation will be referred to as that of the hep. 2 aa.)

Twelve dogs were operated. With no antibiotics given, the first two of them died of liver necrosis, in spite of their having a systematic ramification of intrahepatic arteries. So penicillin was given to the other 10 postoperatively. Two of them (V_{69}, V_{70}) died within 36 hours, and one (V_{35}) on the 3rd day, both of liver necrosis; one (No. 72) died of multiple liver abscesses on the 22nd day. One (No. 68) had its abdomen reopened at the 72nd hour, showing a high degree of necrotic changes in the liver lobes. This died the following day without recovering from the anesthesia, and revealed such a high degree of central necrobiosis or necrosis that the animal would have had a poor chance of survival.

One (No. 57) was found missing (perhaps died) 5 days after ligation; and only the rest 4 survived.

These four were further submitted to the 2nd stage ligation previously mentioned. When reoperated for this purpose 3 to 9 days after the lst ligation, they all showed different degrees of macroscopic and microscopic changes chiefly in the liver edges, just as in those cases whose hep. 3 aa. were ligated. None were found without suffering any changes, but they all well tolerated the 2nd stage ligation until about half a month later they were secrificed to reveal that these changes had been nearly or completely cured.

Plastic injection was performed on 3 of the cases that had died. Although the

plastic pattern as it was did not present a true picture of the anatomy of hepatic circulation systems, some ramification of arterial branches, now replaced by plastics, was clearly recognized in the liver, especially in that of Case (V_{35}) which died 3rd day after the ligation. (Fig. 14) What is more, it was made clear by histological examination that the above 3 or 4 cases had died of massive liver necrosis in spite of antibiotic therapy and presence of arterial branches remaining in the liver (Fig. 14′).

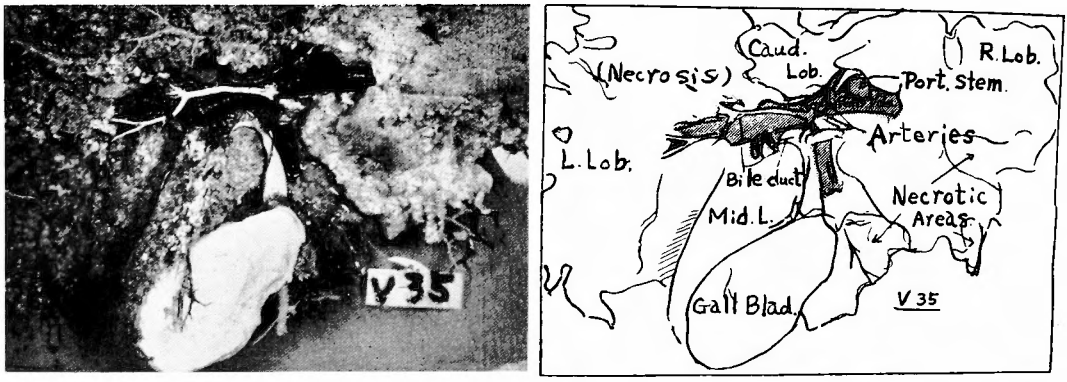


Fig. 14 Case-V_{35} which died from ligation of hep. 2 aa.. Much about the same as the case-V_{39} (Fig. 13)

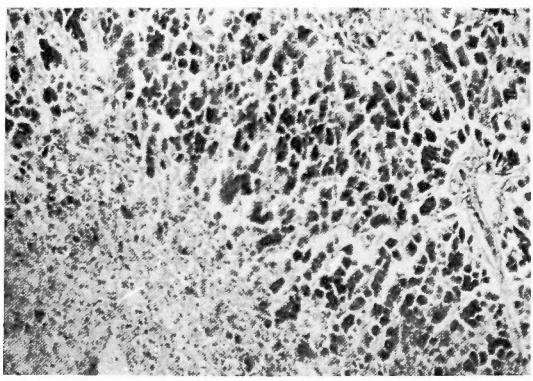


Fig. 14′. Histological picture of the above, case-V_{35}, stained by H-E. (×100.)

Without antibiotics, both of the two dogs with ligation of the hep. 2 aa. died of liver necrosis, while with antibiotics, even if the one that died of liver abscess and the one found missing were not taken its account, 4 Cases out of 8 died of liver necrosis showing 50% of mortality rate. On the other hand, the mortality rate stood at about 54% in the case of ligation of the hep. 3 aa. and about 57% in the case of the hep. 4 aa.. It is very interesting to note here that there is little difference in these 3 figures. (Table 2) More interesting still, if dogs well tolerated the ligation of the c. h. a. and the g. d. a., they all survived the 2nd stage ligation of both the r. g. a. and other arterial collaterals, though suffering quite a high degree of changes in some parts of the liver. This will make us doubtful whether the r. g. a., which has been regarded as an important collateral to the liver, is really so or not.

Table. 2 Mortality of Dogs with Ligation of Hepatic Arteries by Author's Methods.

(with Penicillin Treatment)	Number of Cases	Dead Cases	Mortality
Ligation of Hepatic 3 Arteries	13	7	54%
Ligation of Hepatic 2 Arteries	8	4	50%
Ligation of Hepatic 4 Arteries	7	4	57%

Note: Hepatic 2 Aa. = Common Hepatic A. + Gastro-duodenal A.
Hepatic 3 Aa. = 〃 〃 〃 + 〃 〃 〃 + Right Gastric A.
Hepatic 4 Aa. = 〃 〃 〃 + 〃 〃 〃 + 〃 〃 〃
+ Connecting Arterial Branches (originated from Left Gastric A. or Phrenic A.)

3) Comment

In the above experiment, those cases which died had some arterial blood flown into the liver, just as in those which survived. From this it may be safely concluded that persistence of the liver or survival of the dog does not necessarily depend upon whether or not the dye or plastics, injected from the thoracic aorta after ligation of the chief hep. aa., flow into the liver, or in other words, whether or not some arterial supply to the liver is available by any other route than the ligated main arteries.

IV SUMMARY AND DISCUSSION

Even if the common hepatic artery, the gastroduodenal artery and the right gastric artery of dogs have been ligated, nearly all of them can survive under postoperative administration of antibiotics. The reason for their survival has been thought that some arterial collaterals to the liver work as a substitute for the ligated arteries. GRINDLAY, MANN and BOLLMAN, for example, reported on arterial connecting branches chiefly between the liver and the diaphragm, through which arterial blood flows into the liver, keeping it alive. POPPER and his associates like JEFFERSON, NECHELES and PROFFITT put emphasis on this collateral circulation and averred that so long as it was left intact, dogs with ligation of other arteries survived without antibiotics, whereas when it was completely interrupted, the dogs died no matter however large amounts of antibiotics might be given to them. This view is supported by many in Japan, too.

On the other hand, MARKOWITZ, RAPPAPORT or EZE and others suggested the important role this collateral circulation plays in preventing liver necrosis. Indeed, penicillin given for several days postoperatively can lower the mortality of dogs operated on, and this is, as they assert, because it protects the organ for a week or so, until some arterial collaterals have been developed to supply the liver with sufficient blood to keep it alive.

LAUFMAN et al and WITTER et al reported the existence of anatomical variations of extrahepatic arterial system. They pointed out small vessels in or from the diaphragm, hepatoduodenal ligament, or hepatorenal ligament as possible collateral passages to the liver, and said that the more such vessels existed the higher survival of dogs resulted. Besides, many others like LIVIERATO, HUGGINS, POST and DESFORGAS

referred to such collateral arterial circulations.

On the contrary, some others like TANTURI et al., denying the existence of this collateral circulation from the phrenic arteries in dogs which survived, said that even though there were any small vessels coursing through the hepatoduodenal ligament to reach the liver, they were not enough for dogs operated on to survive the ligation. Although MARKOWITZ et al recognized the presence of arterial collaterals to the liver of dogs operated on, they agreed with TANTURI and others, admitting that the dog's liver survived the loss of its arterial blood supply.

In our clinic, URABE, investigating this point roentogenologically, reported that in dogs which survived, effective arterial collaterals were absent at least within 2 weeks after the ligation.

As was previously stated, especially in such a lobulated liver as the dog's, arterial blood has to come into the liver at its hilus in order to supply each and every lobe systematically. Therfore, any arterial collaterals have either to enter the liver at its hilus, or to anastomose with the existing branches of the proper hepatic arteries at the hilus so that they may supply the whole liver as a substitute for the hepatic 3 arteries that have been ligated. Moreover, they have to course in the liver always with branches of the potal vein, since vascularization of new arteries is next to impossible in a short period after the ligation. And collaterals coming in by any other route could supply only small portions of a single liver lobe.

On the basis of this reasoning, I made the present experiments using the injection method in an attempt to investigate the problem of collateral circulation to the liver after ligation of hepatic arteries of dogs. It was found that the highest possible collaterals are those branches which arise from an arterial network in or near the cardiac part of the stomach and anastomose with the left main branch of the proper hepatic artery. On the one hand, these can be traced back chiefly to the left gastric artery and on the other, they are expected to anastomose with capillary vessels from the neighbouring phrenic arteries. Such being the case, not stereoscopic observation like usual roentogenoscopy may give the impression that the collaterals originate in the inferior phrenic arteries to enter the liver. However, except the above-mentioned collaterals, such connectives from the phrenic arteries to the liver, on the presence of which JEFFERSON, POPPER and others have often insisted, have not been found in the plastic patterns of 63 dogs I injected. Even though those connecting branches I recognized were the same as they had found, they would be too thin to supply the whole liver. And if it was admitted that arterial blood always ran from near the cardiac part of the stomach to the liver, only very little blood could be supplied by way of these connecting branches under normal conditions.

In the present experiment, 4 weeks after ligation one case (V_{45}) was found with a considerably remarkable developement of such collaterals. Indeed, at this late period (4 weeks after ligation) these collateral arteries may well have become worthy of the name, but how much could have been expected of them so soon after the ligation? Were they effective during a short, yet decisive period immediately after the ligation · as short as a dose of penicillin remained effective, yet so decisive that

the fate of the operated dogs depends upon it? On the other hand, even those dogs which were found to have been receiving arterial blood supply to the liver chiefly by this route could not survive in spite of administration of several doses of penicillin.

Since the time of HABERER, POPPER, JEFFERSON and others have emphasized the right gastric artery as an important collateral to the liver. In order to evaluate this opinion, this artery was intentionally left untouched at the time of ligation. The result was that some of the dogs operated upon died of liver necrosis, even when some arterial blood had been flowing into the liver through this route. Then, how could dogs possibly survive with only capillary vessels entering localized, superficial part of the liver where the diaphragm is attached to? How could they live with nothing but those remaining thin vessels leading to the liver in the gastroduodenal ligament, or with nutrient vessels to the walls of the inferior vena cava, the portal vein or the common bile duct?

The present experiment has made it clear that neither by ligation of the hepatic 4 arteries nor by the 2nd stage method of ligation was it possible to interrupt arterial blood supply to the liver completely. Even a more extensive ligation of the hepatic arteries, which has been tried by LAUFMAN or FRASER and others, could not deprive the liver of its entire arterial blood supply.

It has also proved that it is only the hepatic 3 arteries coming in at the hilus, that is, the common hepatic artery, gastroduodenal artery, and right gastric artery that are most closely related with survived of the liver. Whether or not arterial blood comes into the liver by any other route should be considered to have nothing to do with development of massive liver necrosis, nor with survival of the dog operated on. What is more, the results of my present experiment show that the right gastric artery plays no important role inprevention of liver necrosis.

On the other hand, in spite of such an extensive ligation of the chief hepatic arteries that no effective arterial branches might be left uninterrupted in the liver, some cases survived for a long time with the help of antibiotics without showing any marked liver necrosis. This may be proof that the liver can live on the portal blood only without receiving arterial blood.

In normal dogs, however, ligation of the hepatic 3 arteries always leads to lesions in the liver parenchyma due to circulation impediment, regardless of administration of antibiotics. Even though these lesions were reversible, a comparatively high degree of lesions in the cells could not be prevented. So care must be taken when ligation of hepatic arteries is performed under normal condition.

V. CONCLUSION

In the present experiment, in order to investigate the problem of ligation of hepatic arteries and arterial collateral circulation, chiefly coloured plastics were injected into the hepatic vascular systems of dogs through the thoracic aorta, the portal stem and the inferior vena cava. The results are as follows:

(1) Almost complete interruption of arterial blood supply to the liver of dogs

can be done by ligating the common hepatic artery, gastroduodenal artery and right gastric artery. (referred to as the hepatic 3 arteries.)

All dogs deprived in this way of blood supply to the liver died of liver necrosis. Those which were saved by postoperative administration of antibiotics showed some thin intrahepatic arterial branches. These, however, should not be considered enough to account for their survival.

(2) When the hepatic 3 arteries have been ligated, what may be expected to work as chief collaterals to the liver are those small arterial branches coursing from the left gastric artery (or the phrenic arteries) through the cardiac part of the stomach to anastomose with the left main branch of the proper hepatic arteries. Even if as many arteries as possible, including these collaterals, were ligated, it resulted in much the same mortality of dogs operated on as otherwise.

(3) All dogs which died of liver necrosis in spite of postoperative antibiotic therapy revealed some arterioles remaining in the liver. Some of the dogs had even a systematic ramification of considerably effective arterial branches.

(4) When the common hepatic artery and gastraduodenal artery alone were ligated, with the right gastric artery and other collaterals intentionally left intact, some dogs died of liver necrosis in spite of postoperative administration of antibiotics. If dogs were able to survive this method of ligation, they could also survive the added ligation of the other arteries.

(5) Even when such an extensive ligation of hepatic arteries were performed that no effective arterial blood might flow into the liver, some cases survived with no remarkable changes in the liver.

From the above findings it may safely be concluded that it is upon the common hepatic artery and gastroduodenal artery that the dog's liver depends for its survival; that no other arteries to the liver decide the fate of the dogs operated on; and that even if the liver receives no arterial supply, it can live and function well only with the portal venous blood.

In concluding my report, I wish to express my deepest gratitude both to Dr. CHISATO ARAKI, professor of Kyoto Univ. and Dr. ICHIO HONJO, assist. professor of the same for their kind, continuous guidance, encouragement and supervision. Pertaining to histological findings, I am greatly indebted to Dr. YOSHIHIRO HAMAJIMA, assist. professor of the pathological division, Kyoto Univ. Medical School for his kind guidance.

REFERENCES

1) Chau, A. Y. S., Goldbloom, V. C. & Gurd, F. N.: Clostridial Infection as a Cause of Death after Ligation of the Hepatic Artery. Arch. Surg., **63**, 390, 1951.
2) Child, G. C.: The Hepatic Circulation and Portal Hypertension. Philadelphia, Saunders, 1954.
3) Desforgas, G., Campbell, A. J. & Robbins, S. L.: Hepatic Arterial Ligation for Portal Hypertension. Ann. Surg., **137**, 507, 1953.
4) Eguchi, S. & Daito, Y.: A New Method of Casting of the Canalicular System and Mounting Sections with Acrylic resin. Acta Scholae Med. Gifu., **2**, 203, 1954.
5) Eze, W. C.: Cause of Survival of Dogs without a Hepatic Artery. Arch. Surg. **65**, 684, 1952.
6) Fitts, W. T., Scott, R. & Mackie, J.: Effect of Antibiotics on Ligation of Hepatic Artery.

Cites by Grant et al. Nov. 1950.
7) Fraser, D., Rappaport, A. M., Vuylsteke, C. A. & Colwell, A. R.: Effects of the Ligation of Hepatic Arteries in Dogs. Surg. **30**, 624, 1951.
8) Grant, J. L., Fitts, W. T. & Ravdin, I. S.: Aneurysm of the Hepatic Artery. Report of Two Cases and a Consideration of Surgical Treatment. Sur. Gyn. & Obst. **91**, 527, 1950.
9) Grindlay, J., Mann, F. C. & Bollman, J. L.: Effect of Occulsion of the Arterial Blood Supply to the Normal Liver. Arch. Surg. **62**, 806, 1951.
10) Grindlay, S.: Experimentally Produced Alteration of Hepatic Blood Flow. Arch. Surg. **66**, 562. 1953.
11) Haberer, H.: Experimentelle Unterbindung der Leberarterie. Arch. für Klin. Chir., **78**, 557, 1905.
12) Honjo, I.: Liver Cirrhosis and Hepatic Circulation. Jap. Clin. "Nihon Rinsho" in Jap., **15**, 1069, 1957.
13) Huggins, C. & Post, J.: Experimental Subtotal Ligation of the Arteries Supplying the Liver. Arch. Surg., **35**, 878, 1937.
14) Ikegami, K.: The Vasological Study on the Gastro-enterostomy by Means of Plastic Injection Method. Arch. für Jap. Chir., **28**, 467, 1959.
15) Imanaga, H.: Operation and Hepatic Lesions. "Sogo Rinssho" in Jap., **7**, 1716, 1958.
16) Imanaga, H.: Studies on Ascites Formation and its Treatment. J. Nagoya Med. "Nagoya Igaku" in Jap., **77**, 562, 1959.
17) Jefferson, N. C., Proffitt, M. M. & Necheles, H.: Collateral Arterial Circulation to the Liver. Am. J. Physiol. **167**, 800, 1951.
18) Jefferson, N. C., Proffitt, M. M. & Necheles, H.: Collateral Arterial Circulation to the Liver of the Dog. Surg. **31**, 724, 1952.
19) Laufman, H., Ross, A., Bernhard, V. M., Bourdeau, R. V., Furr, W. E. & Douglass, J. C.: Graded Hepatic Arterial Ligations in Experimental Ascites. Surg. Gyn. & Obst, **96**, 409, 1953.
20) Markowitz, J., Rappaport, A. M. & Scott, A. C.: Prevention of liver Necrosis Following Ligation of Hepatic Arteries. Proc. Soc. Exper. Biol. & Med., **70**, 305, 1949.
21) Markowitz, J., Rappaport, A. M. & Scott, A. C.: The Function of the Hepatic Artery in the Dog. Am. J. Digest. Dis. **16**, 344, 1949.
22) Markowitz, J. & Rappaport, A. M.: The Hepatic Artery. Physiol. Rev. **31**, 188, 1951.
23) Matsuzaki, I.: A Study of Ligation of Hepatic Artery. J. Nagoya Med. "Nagoya Igaku" in Jap., **69**, 640, 1955.
24) Nagazawa, N. & Yamashita, M.: Three-dementional and Microscopic Observation of Healthy or Tubercular Lungs by Means of Plastic Injection Methods. "Kekkaku Kenkyu" in Jap., **8**, 54, 1952.
25) Nishikawa, Y.: Experimental Study of Schistosomiasis Japonica. Morphological Findings of Hepatic Vascular Systems by Means of Plastic Injection Method. Jap. Red. Closs Med. J., "Nisseki Igaku" in Jap., **9**, 41, 1955.
26) Popper, H. L., Jefferson, N. C. &Necheles, H.: Interruption of All Arterial Blood Supply to the Liver, Not Compatible with Life. Am. J. Surg. **84**, 429, 1952.
27) Popper, H. L.: Ligation of Accidentally Torn Hepatic Artery. Am. J. Surg. **85**, 113, 1953.
28) Popper, H. L., Jefferson, N. C. & Necheles, H.: Liver Necrosis Following Complete interruption of Hepatic Artery and artial Ligation of Portal Vein. Am. J. Surg. **86**, 305, 1953.
29) Popper, H. L., Jefferson, N. C. & Necheles, H.: Survival of Dogs After Partial or Total Devascularization of the Liver. Ann. Surg. **140**, 93, 1954.
30) Rappaport, A. M.: Experimental Ishemia of the Liver and Hepatic Coma. Transactions of the 10th Conf. p146. New York, May, 1951.
31) Ravdin, I. S. & Vars, H. M.: Factors Influencing Liver Injury and Liver Repair. Ann. Sur. **132**, 362, 1950.
32) Rienehoff, W. F.: Ligation of the Hepatic and Splenic Arteries in the Treatment of Portal Hypertension with a Report of Six Cases. Bull. J. Hopk. Hosp., **88**, 368, 1951.
33) Segall, H. N.: An Experimental Anatomical Investigation of the Blood and Bile Channels

of the Liver. Surg. Gyn. & Obst., **37**, 152, 1923.
34) Takeuchi, K.: Morphological Study of Hepatic Vascular Systems. Jap. J. Constitut. Med., **22**, 46, 1957.
35) Tanturi, C., Swigart, L. & Canepa, J.: Prevention of Death from Experimental Ligation of the Liver (Hepatic Proper) Branches of the Hepatic Artery. Sur. Gyn. & Obst. **91**, 680, 1950.
36) Toyoshima, H.: Surgical and Anatomical Studies on Liver Structure by Injection of Accryric resin. Arch. fur Jap. Chir., **23**, 476, 1954.
37) Toyoshima, H.: How to Make Plastic Patterns of Hepatic Circulation System and Bile Duct. Resp. & Circulat. "Kokyu to Junkan" in Jap., **4**, 548, 1956.
38) Urabe, H.: Experimental Study of the Interruption of the Arterial Blood Flow to the Liver. Arch. für Jap. Chir., **28**, 1112, 1959.
39) Witter, J. A. & First, M.: Ligation of the Hepatic and Splenic Arteries for Advanced Periportal Cirrhosis. Surg. **33**, 663, 1953.
40) Yamagishi, K.: Intrahepatic Vascular System and Course of Bile Duct in Man and Animals. J. Exp. Gastroenterolog. "Jikken Shokakibyo Gakkai Zashi" in Jap., **4**, 984, 1929.

和文抄録

犬に於ける肝動脈遮断と動脈性副血行路の問題

京都大学医学部外科学教室第1講座（指導：荒木千里教授）

石　黒　　稔

犬に於て，総肝動脈・胃十二指腸動脈，及び右胃動脈の3動脈を遮断した場合，術後抗生物質の使用に依り大半の犬がよく生存を続けうる理由として従来から，主として之等3動脈以外の肝に到る動脈路が代償するのでないかと憶測説明されて来た．そこで私は，約90例の成熟犬を実験に用い，その63例に於て肝内外血管系へ墨汁或は着色合成樹脂を注入し，又，その鋳型標本について之等の副血行路の存在と，肝への動脈路を遮断された後に於ける致死的な肝壊死発生との関係について追究し主として次の結果を得た．

1) 犬に於て，肝へのあらゆる動脈血をすべて完全に遮断することは困難であるが，総肝動脈・胃十二指腸動脈及び右胃動脈を遮断すれば，肝に到る動脈血を殆ど遮断することが出来る．之等の遮断を行えば，全例広汎な肝壊死発生に依り死亡するが，遮断後抗生物質を使用することに依り死を免れた犬に於て，術後幾月かを経過した屠殺時に，爾他の経路から尚若干の動脈性微細枝の肝内分枝の残存を認め得ても，之等は何れもその生存を理由づけうるに足るものではない．

2) 上記三動脈を遮断した場合に，主な副血行路として横隔膜からの肝への分枝が多くの先人達に依つて指摘され且つ重視されている．然し私の観察に依れば，之等が肝に対し直接与える分枝は皆無に等しく，唯，こゝに比較的有力な経路として，左胃動脈の枝が胃噴門部及び横隔膜食道孔附近で，横隔膜動脈の枝と共に互に吻合し，之等から2，3の細小枝が既存の固有肝動脈左枝に連絡するものをほゞ全例に認めた．蓋し，犬の如き分葉肝に於ては特に，肝に対する動脈路が，各肝葉全体に系統的且つ瀰漫性に給血せんとする場合は，あくまで肝門部よりの給血に俟たねばならない．従つて動脈性副血行路が遮断された三動脈に代つて肝全体を養いうる為には，それが肝門部より入るか，或は肝門部の既存の固有肝動脈枝に連絡しうるもののみが意味があると考えられる．この意味に於て上述の胃噴門部よりの連絡枝こそは，肝門部よりの主動脈路を可及的広汎に遮断した後に於ける有力な副血行路生成の可能性として挙げられる．然し正常時はもとより，術直後の肝壊死発生に重大な関係を有する短期間内に於ては，この経路も尚極めて微々たるものにすぎない．更に又，この経路をも含めて可能な限りの動脈路を遮断した私の実験に於ても，その死亡率に上述の場合と特に有意の差を認めない．

3) 遮断後抗生物質の使用にも拘らず，肝壊死に依り死亡した犬に於ても又，全例に若干の動脈性細小枝の肝内残存から認められ，中には相当有力な動脈枝の系統的肝内分枝が見られるものすらあつた．

4) 意識的に右胃動脈及び爾他の副血行路を残して総肝動脈・胃十二指腸動脈のみを遮断し，且つ抗生物質を使用しても肝壊死を発生して死亡する例が，上述の遮断法に依るものとほゞ同率に認められる．又この2動脈のみの遮断後生存し得た犬は，更に右胃動脈及び他の副血行路の遮断を追加してもよく耐え得た．このことは又，従来肝への有力な副血行路と見做されてきた右胃動脈の意義に関し再考を要するものと考えられる．

5) 肝への動脈路を可能な限り広く遮断し，肝内に有効な動脈血流入が見られなくても，尚肝実質に著明な変化なく生存を続け得た例を得ることが出来た．

以上の事実から，肝壊死発生に最も重大な関係を有するものは，総肝動脈及び胃十二指腸動脈の2者のみであり，之以外の経路から肝に到る動脈枝残存の有無は，主動脈路遮断後の犬の生死を左右する因子ではなく，又仮令有効な動脈血の流入がなくとも，肝は門脈血行のみに依つて充分存続し得，且つその機能を全うしうるものと考えられる．